results were impressive. Some 44 percent experienced a greater than 75 percent reduction in seizure frequency; 25 percent were slightly improved; 31 percent were considered treatment failures. DPA is most effective in generalized and least effective in focal epilepsies. It has been useful in both adults and chlidren, when administered alone and with other anticonvulsants. It should be noted that valproate is FDA-approved only for use in absence seizures.

DPA probably will become the drug of choice in myoclonic and akinetic seizures, where only clonazepam has previously been helpful. Valproate is a welcome addition to the treatment of infantile spasms, for which adrenocorticotropic hormones and ketogenic diets are therapies of desperation. Its role in any but intractable tonic-clonic seizures is limited, for the present author, by its short half-life which requires multiple daily doses, and by possible toxicity. Refractory status epilepticus may respond to administration by gavage or by rectal suppository.

DPA has been advocated for the treatment of typical and atypical absence attacks (staring spells with or without automatisms). It is undoubtedly effective in these cases, but should probably be restricted to intractable cases, pending elucidation of toxicity. For prophylaxis of febrile convulsions, DPA has the advantage of less sedation than traditional therapies, but the same reservations apply.

Valproate is rapidly and completely absorbed after oral administration. Serum levels peak at one to four hours. In animals, DPA reaches the brain within minutes. The drug is highly (90 percent to 95 percent) protein-bound, but widely distributed. It crosses the placenta and enters (though at lower concentrations than primidone) human breast milk. After omega-hydroxylation and glucuronide conjugation by the liver, the drug is mostly excreted in the urine. Hepatic dysfunction is a relative contraindication to its use. Plasma half-life is 8 to 15 hours: less in children and in patients receiving phenytoin, phenobarbital, primidone, carbamazepine or ethosuximide. American investigators have used up to 50 mg per kg of body weight in adults. Standard adult schedules start with 250 mg twice a day, increasing at three to seven day intervals until control, toxicity or a maximum dose of 2,400 to 2,600 mg is achieved. Use of other anticonvulsants is monitored but maintained; maximum benefits from DPA may be deferred two or more weeks. The mechanism of action is unknown.

In summary, valproate is a major addition to the anticonvulsant armamentarium. Alone and with other drugs, it will find its place as experience with its use and toxicity grows.

ROBERT I. PFEFFER, MD

REFERENCES

Simon D, Perry JK: Sodium di-N-propylactate (DPA) in the treatment of epilepsy: A review. Epilepsia 16:549-573, Nov 1975

Pinder RM, Brogden RN, Speight TM, et al: Sodium valproate: A review of its pharmacological properties and therapeutic efficacy in epilepsy. Drugs 13:81-123, Feb 1977

Willmore LJ, Wilder BJ, Bruni J, et al: Effect of valproic acid on hepatic function. Neurology 28:961-964, Sep 1978

Meralgia Paresthetica

THE SYNDROME of numbness, tingling, burning or pain in the anterolateral thigh was first described in 1885 and later named meralgia paresthetica (*meros,* thigh; *algos,* pain). This mononeuropathy of the lateral femoral cutaneous nerve is a rare cause of severe pain; however, the paresthesias associated with this entity are more common than previously recognized and the syndrome is frequently misdiagnosed.

Typically, meralgia paresthetica is first seen in middle age and is a sporadic affliction. Nevertheless, the duration of symptoms before diagnosis may be quite variable, ranging from weeks to many years. There is a frequent association with obesity and the condition is reported to be bilateral in 8 percent to 12 percent of the cases.

It is thought that the long course and unique anatomical relationships of the lateral femoral cutaneous nerve make it particularly susceptible to mechanical forces. The nerve exits the lumbar plexus and passes beneath the psoas muscle, curving and descending to the level of the anterior superior iliac spine. It is here that the nerve angles sharply downward and passes under or through attachments of the inguinal ligament. Finally, the nerve pierces the overlying fascia and enters the anterolateral thigh.

The cause of meralgia paresthetica is occasionally identifiable. However, most cases are idiopathic. Reported antecedents of the syndrome include local trauma, pregnancy, intrapelvic disease, seat belts, braces, local tumors, disc lesions and prolonged hip extension. In a recent anatomical study, Edelson and Nathan found that in 51 percent of unselected adult autopsy cases there was a significant enlargement at the ligamentous stress point. The authors postulate that this pseudoganglion results from mechanical irritation and

may play a role in otherwise idiopathic cases of meralgia paresthetica.

Treatment depends largely upon a patient's history. In many persons relief is obtained from benign neglect or weight reduction, while in others there is response to local injections of lidocaine or steroids. In selected cases surgical manipulations, such as decompression, lateral transposition or complete transection of the nerve, have been employed successfully.

THOMAS J. CHIPPENDALE, PhD
Fourth-Year Medical Student

REFERENCES

Edelson JG, Nathan H: Meralgia paresthetica: An anatomical interpretation. Clin Orthop 122:255-262, Jan-Feb 1977

Kitchen C, Simpson J: Meralgia paresthetica: A review of 67 patients. Acta Neurol Scand 48:547-555, 1972

Ecker AD, Woltman HW: Meralgia paresthetica: A report of 150 cases. JAMA 110:1650-1652, 1938

Rehabilitation of Patients With Stroke

PATIENTS WITH A completed stroke and moderate to severe functional deficits can be rehabilitated and returned home, rather than sent to a chronic care facility.

A patient with an acute stroke who is not independent enough to return home when medically stable should ideally be transferred to the intensive, multidisciplinary, inpatient program of a regional rehabilitation center that treats at least 200 patients each year. Duration of inpatient rehabilitation ranges from 4 to 10 weeks, depending on the severity of neurologic and functional deficits and the degree of independence in activities of daily living necessary to live at home or in a board-and-care facility.

Factors that have negative effect on outcome of rehabilitation include long onset before admission to a specialty center, severe perceptual or cognitive dysfunction, and poor motivation. Severe weakness, sensory loss, dysphasia or visual field loss, and age or underlying medical problems may slow progress, but they are often overcome. These are not contraindications to a trial of formal rehabilitation.

With intensive inpatient therapy, about 80 percent to 85 percent of survivors of stroke will return home. About 80 percent will be independent in mobility and 60 percent will be independent in self care. Improved functional gains, especially for dressing, bowel and bladder function, transfers and walking may develop with therapy even one year after onset of stroke, and are maintained by most patients. Aphasic patients make significant gains with formal speech therapy compared with untreated aphasic patients; comprehension tends to improve more than expressive tasks. No single therapeutic modality seems more effective than another. Therefore, an eclectic approach to deal with a patient's particular neurologic and functional disability seems warranted.

Unfortunately, the natural history of recovery of a stroke patient with a particular set of symptoms, signs, laboratory studies, and findings by angiography and computerized tomography has not been available. Thus the value of specific neuromedical and rehabilitative interventions will be uncertain, until similar patients are compared prospectively. The national Stroke Data Bank Pilot Project has recently begun to address this problem.

BRUCE H. DOBKIN, MD

REFERENCES

Feigenson J: Stroke rehabilitation: Effectiveness, benefits, and cost. Stroke 10:1-4, Jan 1979

Gresham GE, Fitzpatrick TE, Wolf PA, et al: Residual disability in survivors of stroke—The Framingham study. N Engl J Med 293: 954-956, Nov 6, 1975

Basso A, Capitani E, Vignolo L: Influence of rehabilitation on language skills in aphasic patients. Arch Neurol 36:190-196, Apr 1979

Management of Cluster Headaches

THE MAIN FEATURES of cluster headaches include extremely severe unilateral pain which is most often periorbital, of short duration (20 to 90 minutes), and associated with ipsilateral ptosis, miosis, conjunctival injection and rhinorrhea. In many patients there is a periodic "clustering" of headaches followed by an extended remission period. This is termed episodic cluster. A chronic type of cluster headache has recently been identified. In this category typical cluster headaches occur without any remission period. A rare subgroup of the chronic variety has recently been described as chronic paroxysmal hemicrania (CPH), based on its unique therapeutic response. This type of clinical classification is useful in choosing the appropriate drug in a disorder that is difficult to treat.

In a patient with episodic cluster headaches, especially with a clock-like regularity to the attacks, 1 to 2 mg of ergotamine tartrate administered orally an hour before the anticipated time of the headache provides the best prophylactic therapy. Most often a bedtime dose is sufficient to prevent headaches at night. The timing of the headache may occasionally change after initiating this form of treatment, and one may have to "chase the headache" with ergots. If the timing is erratic